owing to the greater strength of bone over cartilage. Starting from an almost entire lack of bone in the first year of life, one will notice that these bones increase in number and in size until finally the almost fully developed wrist is attained at about the age of fourteen to fifteen years. This assumes that the quality of the bone tissue is also normal. This also necessitates a qualitative study in the future of bone tissue as well as bone size.

It is thus seen that there is a complete series of stages of development of the wrist from infancy to adolescence, showing in this way different stages of qualitative and quantitative strength, each of which should be equal to the endurance called for by certain kinds of mental fatigue as well as manual labor.

COMPRESSION OF THE PULMONARY VEINS, THE PRESSURE FACTOR IN THE ETIOLOGY OF CARDIAC HYDROTHORAX.[1]

By George Fetterolf, A.B., M.D.,
DEMONSTRATOR OF ANATOMY IN THE UNIVERSITY OF PENNSYLVANIA; LARYNGOLOGIST TO THE WHITE HAVEN (PENNSYLVANIA) SANATORIUM AND THE HENRY PHIPPS INSTITUTE, PHILADELPHIA,

AND

H. R. M. Landis, A.B., M.D.,
VISITING PHYSICIAN TO THE WHITE HAVEN (PENNSYLVANIA) SANATORIUM AND THE HENRY PHIPPS INSTITUTE; DEMONSTRATOR OF MEDICINE IN THE JEFFERSON MEDICAL COLLEGE, PHILADELPHIA.

(From the Laboratory of Anatomy of the University of Pennsylvania and the Jefferson Medical College Hospital.)

The hydrothorax that sometimes accompanies heart disease has been recognized for many years. Why it occurs in some cases and not in others is not clear, for it is certain, as both Flint and Da Costa[2] long since remarked, that the phenomenon bears no constant relation to the extent of the cardiac disease. "Not only are these lesions (cardiac) marked in cases in which dropsy has not occurred, but dropsy occurs in other cases in which the lesions are comparatively slight" (Flint[3]).

In seeking an adequate explanation for the occurrence of these effusions we have concerned ourselves chiefly with the effects, the mechanical effects, arising from pressure exerted by the dilated heart. We recognize fully that before an effusion can occur the presence of factors other than pressure may be necessary, for example, toxic, vasomotor, bacterial, or metabolic influences, or a hydremic condition of the blood. Krehl[4] states in this connection: "Certain patients suffering from heart disease in the state of broken compensation show a watery condition of the blood, both the specific gravity and the proportion of proteids in the blood being diminished (Grawitz). A weakness of the right ventricle is especially apt to give rise to such a thing in the blood. . . . This hydremia occurs in a comparatively small proportion of all cases of broken compensation; but where it does occur it is quite usual for it to disappear with any improvement in the circulation."

---

[1] Read at a meeting of the Pathological Society of Philadelphia, May 27, 1909.

[2] Medical Diagnosis, eighth edition, p. 422.

[3] Diseases of the Heart, second edition, 1870, p. 37.

Any one of those agents, however, which affect the composition of the blood will fail to account for the fact that these effusions have a marked preference for one or the other side of the chest, usually the right. That pressure alone will cause the transudate is improbable, but it is certainly a factor of the greatest importance. Another point is that the pressure must be from dilatation, as it has long been recognized that hypertrophy of the heart by itself is incapable of producing an effusion, and that dilatation, with or without valvular lesions, is an essential feature.

Recognizing that dilatation of the heart was necessary, most observers contented themselves with the explanation that a transudation resulted from venous stasis, either alone or in association with impoverishment of the blood. In recent years, however, the azygos-vein theory has been much in favor, largely as a result of papers published by Steele[5] and Stengel.[6] Their explanation of these effusions, and particularly of the right-sided preponderance, is that the dilated right heart by extension upward exerts pressure on the root of the right lung and thus indirectly pinches the azygos major vein as it curves over the root of the right lung to enter the superior vena cava. Baccelli,[7] in 1863, first called attention to this possibility, although his explanation differed somewhat from that of Steele and Stengel. His idea was that the enlarged heart, by dragging the superior vena cava downward, carried with it the vena azygos major, thus drawing it tightly around the root of the lung and causing it to be compressed.

What has commended this theory to many is the fact that the vena azygos major receives as tributaries the intercostal veins of the greater portion of the right chest, vessels which drain the major portion of the right costal pleura. The natural assumption has been that with compression of this vein in its upper part there would result a venous stasis on the right side which would lead to a transudation. The theory has gained additional strength from the fact that these effusions are so frequently right-sided, or, when bilateral, greater on the right side. When the condition was bilateral the smaller, left-sided effusion was explained on the basis that the vena azygos minor, which drains less of the left chest than does the major of the right, and empties into the latter at the eighth or ninth thoracic vertebra, would become affected only secondarily and to a slighter extent.

---

[4] Clinical Pathology, American edition, 1905, p. 176.

[5] University Medical Magazine, 1897, Journal of the American Medical Association, October 1, 1904.

[6] University of Pennsylvania Medical Bulletin, 1901.

[7] Quoted by Steele, Jour. Amer. Med. Assoc., October 1, 1904.

Every concrete theory hitherto advanced to explain the condition has been based on the assumption that the fluid is derived from the parietal pleura; and, of the vessels of this part of the membrane, the azygos veins have been held by most writers to be the source of the fluid. To this explanation five objections can be raised: (1) Only about two-thirds of the parietal membrane is drained by the azygos veins; (2) the collateral anastomoses of the azygos veins are so free and so numerous that, in the event of pressure, competent by-paths would soon be established and carry away any excess of fluid in the azygos radicles; (3) the vena azygos minor, emptying into the major, is subject to the same influences as the latter, and therefore the effusion should always be bilateral; (4) it is anatomically impossible for the heart, either directly or indirectly, to exert pressure upon the azygos major vein; (5) it does not explain purely left-sided effusions.

1. First, as to the azygos veins draining only about two-thirds of the parietal pleura. Like all serous membranes, the pleura consists of two portions, a parietal and a visceral, and the circulation in the two differs greatly, each receiving and discharging blood in vessels which belong to the structures with which the membrane is in contact. An example of this principle is afforded by the pericardium, of which the vessels of the parietal layer are derived from the thoracic aorta and the internal mammary, while those of the visceral layer or epicardium are branches of the coronaries. The pleura has a similar arrangement, but for the present the vessels of only the parietal layer will be considered. The arteries are derived from the superior and aortic intercostals, the internal mammary, the mediastinal, the œsophageal, the bronchial, and the phrenic, and the return circulation is through corresponding veins.

The most important thing to have clear in this connection is the vessels into which these veins drain. The intercostals vary somewhat at their vertebral ends, but the average arrangement is for the first intercostal on both right and left sides to empty into the innominate, while the second, third, and fourth on the right side form a trunk which opens into the vena azygos major, and the remainder on this side open separately into the same vessel. On the left side the second, third, and some times the fourth form a trunk, the superior intercostal vein, which opens above into the innominate and is connected with the left upper azygos vein. The intercostals from the fifth to the eighth on the left side form the left upper azygos and open into the azygos major or minor, while the remaining intercostals open individually into the azygos minor. At their sternal ends the upper nine or ten intercostals of both sides open freely into the internal mammary, or its musculophrenic tributary, the lower two or three having no such termination.

Of the remaining veins of the parietal pleura, the internal mammaries empty into the innominate, the mediastinal into the same vessel, the œsophageal into the azygos and also into the inferior thyroid and the gastric, the bronchial into the azygos, the inferior phrenic into the inferior vena cava, and the superior phrenic into the left innominate. The cervical pleura drains into the innominate, the posterolateral part of the right costal pleura into the azygos major, the upper third of the posterolateral part of the left costal pleura into the innominate above and the azygos minor below, and the lower two-thirds into the azygos minor. The anterior part of the costal pleura empties its blood into the internal mammaries, the mediastinal pleura into vessels which eventually reach the innominate and the portal, and the diaphragmatic pleura into veins which are tributary to the innominate and the inferior vena cava.

Analysis of this summary shows that the venous drainage of the parietal pleura is into widely separated vessels, and consists of veins which open into the azygos major and minor, the innominate, the inferior thyroid, the gastric, and the inferior vena cava. The deduction to be drawn from this is that while the azygos veins are of importance in carrying away the blood from the parietal pleura, only a portion of the membrane is drained by this system, perhaps not more than two-thirds.

2. Second, as to the numerous and wide anastomoses of the azygos veins. In order to render clear this portion of the discussion, it is perhaps advisable to give a brief resume of the tributaries and anastomoses of the azygos veins. The tributaries of the azygos major are the lumbar, the right intercostals with the exception of the first, the œsophageal, the posterior mediastinal, the venous plexus around the thoracic aorta, the posterior pericardiac, the bronchial, and the azygos minor. In connection with these vessels it is important to know how extensive and how practicable are their peripheral anastomoses. The azygos major begins just below the diaphragm, and at this point anastomoses with the right ascending lumbar vein, which connects it with the four right lumbar veins and, through the lowest of these, with the iliolumbar or the common iliac vein. The azygos minor has the same connections on the left side, and by these anastomoses a ready side track into the inferior vena cava is established.

As regards the intercostals, "In the middle portion of their course the upper six or seven veins give off branches, the costo-axillary veins, which ascend toward the axilla and open into either the long thoracic or the thoracico-epigastric vein and so into the axillary, and, as it approaches the vertebral column behind, each vein receives a dorsal branch which accompanies the spinal branch of the intercostal artery and returns the blood from the skin and muscles of the back and also from the spinal column and its contents, this drainage being by means of a spinal branch which connects with the intervertebral veins."[8] "Laterally, at each intervertebral foramen the internal (spinal) plexuses send branches out from the spinal canal along the nerve trunks, and by means of these intervertebral veins, which have the form of plexuses at their origin and receive communicating branches from the external vertebral plexuses and from the veins of the spinal cord, the internal plexuses pour their blood into the vertebral, intercostal, lumbar, and lateral sacral veins, the connection with the intercostals being through their rami spinales."[9] In addition to these anastomoses the upper nine or ten intercostals open anteriorly into the musculophrenic or internal mammary veins. It is evident, therefore, that obstruction to the flow of blood through the intercostals would result in the blood seeking and finding other courses, which are many and wide—the axillary, the vertebral, the lumbar and the lateral sacral.

The œsophageal veins anastomose with the inferior thyroid veins above and the gastric below. There are thus afforded paths into the innominate and the superior vena cava by way of the inferior thyroids and into the portal by way of the gastric. The posterior mediastinal veins open into the left innominate, and the destination of the plexus around the aorta is probably into the azygos and œsophageal veins. The posterior pericardiac veins anastomose freely with the anterior pericardiac, which open into the internal mammary, and through these a path is established into the left innominate or into the superior vena cava. The bronchial veins anatomose in the lung with some of the radicles of the pulmonary vein and thereby afford a wide open channel for any dammed back blood. The vena azygos minor (vena hemiazygos) begins in the left upper abdomen and enters the thorax through the left crus of the diaphragm. Thence it ascends at the left side of the thoracic aorta and at the level of the eighth or ninth thoracic vertebra crosses to the left beneath the thoracic duct and thoracic aorta and empties into the vena azygos major. The tributaries of this vessel are the ascending lumbar vein, the left lower four or five intercostals, the œsophageal, the posterior mediastinal, and occasionally the left upper azygos. The peripheral anastomoses of these vessels are, with the exception of the left upper azygos, the same as have been described in connection with the azygos major.

[8] Piersol's Human Anatomy, p. 896.

[9] Ibid., p. 898.

The left upper azygos vein (vena azygos minor superior, or accessory hemiazygos) begins at the second intercostal space on the left side and descends to the left of the middle line. At about the eighth thoracic vertebra it crosses to the right posterior to the aorta and œsophagus, to empty into the azygos major. Sometimes it empties into the vena azygos minor as the latter curves toward the right, and even when it empties into the azygos major it is frequently connected directly with the azygos minor. Its tributaries are the upper seven or eight intercostal veins, the left posterior bronchial veins, and a twig which connects it with the left innominate. The potentialities of these have been indicated in the description of the azygos major.

It would seem from this summary that the possibilities of a wide and ready collateral circulation through many by-paths, some leading into the superior and some into the inferior vena cava, are so great that it is almost unthinkable that any degree of obstruction could be sufficient to cause leakage into the pleural sac. In this connection it should be remembered that the pleura and its vessels are constantly subjected to a suction or negative pressure equivalent to 6 mm. of mercury, and must, therefore, be stronger and better equipped to resist pressure than any others in the body. Were this not so the pleural cavities of everyone presenting favorable blood conditions would fill with fluid until the pressure in the sac would be equal to the pressure within the lung.

In connection with the above resume of the azygos anastomoses it is interesting to note that Broadbent,[10] in referring to the pressure of aneurysms on the venous trunks, states that "there may be pressure on the azygos vein which passes up behind the root of the lung to open into the superior vena cava just before it enters the pericardium. This does not appear to give rise to any characteristic physical signs or symptoms, probably on account of the free anastomosis with the lumbar veins, which enables the blood to return without difficulty via the iliac veins and the inferior vena cava." This anastomosis mentioned by Broadbent is obviously but one of many.

3. Third, as to the vena azygos minor being subject to the same influences as the major. It will be remembered that the former vein, after receiving the left upper azygos, the lumbar, and the lower intercostal veins, crosses the vertebral column and empties into the azygos major. Being a tributary to the latter and the largest branch of the azygos tree, it must of necessity be subjected to the same pressure influences. Granting that obstruction at the azygos arch is responsible for these right-sided transudates, it is difficult to understand why the effusion should ever be limited to one side, especially the right, since stasis would probably first be manifest at the

---

[10] Heart Disease, fourth edition, p. 445.

greatest distance from the point of pressure, that is, in the radicles of the vena azygos minor.

4. Fourth, as to the possibility of the heart exerting pressure upon the azygos major vein. In order to discuss this intelligently, a review of the course and relations of the vein is essential. The vena azygos major (vena azygos) begins in the right upper abdomen and passes into the thorax either through the aortic opening in the diaphragm or between the median and intermediate portions of the right crus. It ascends in the posterior mediastinum to the right of the median line, having behind it the right intercostal arteries and the bodies of the thoracic vertebræ. To its left lie the aorta, the œsophagus, and the thoracic duct. On reaching the level of the seventh thoracic vertebra it inclines slightly to the right, and at the level of the fifth or fourth bends forward and passes between the mesial surface of the right lung and the right aspect of the trachea at or just above the obtuse angle (155 degrees) at which the right bronchus leaves the trachea. Either at or posterior to the plane of the anterior surface of the trachea it enters the superior vena cava just above the point at which the pericardium leaves the latter vessel. A number of measurements taken in the dissecting room of the University of Pennsylvania showed this point of entrance to be about 6 cm. above the entrance of the superior cava into the right auricle. The terminal portion of the vessel, as it arches over the right bronchus, is called the "azygos arch." Valves, usually to the number of four, are present in the vessel, but they are frequently unpaired and usually incompetent. In 22 per cent. the valves are absent.[11]

To justify the statement that the heart cannot press on the azygos major vein it is necessary to review briefly the anatomical relations at the roots of the lungs. These roots are made up of the bronchus, the pulmonary and bronchial vessels, nerves, lymphatics, and areolar tissue. The superior vena cava descends anterior to the root of the right lung to terminate in the upper portion of the right auricle, and this upper part of the auricle lies in an anterior relation to the root, an important fact, of which use will be made later. After leaving the vertebral column to course forward the azygos major passes in an arciform manner over the right pulmonary root to empty into the superior vena cava about 6 cm. above the termination of the latter in the right auricle. A dilated right heart, to exert any pressure on the azygos arch, would necessarily displace upward and backward the pulmonary veins, the bronchus, the pulmonary arteries, and all the minor elements in the lung root. To realize that this is an impossibility one has but to study the actual preparation made in a body in which the viscera have been hardened in situ, and to take into consideration not only the intrinsic strength and resistance of these structures, but also their firmness of attachment. The ligamentum latum pulmonis alone, a firm reflection of the pleura connecting the lung root with the diaphragm, would be a potent factor in restraining any upward excursion of the bronchus and its associated structures. Study of the accompanying figures (Figs. 1 and 2) should convince one that the pressure claimed to be possible is out of the question. Theoretically there might be some direct pressure exerted by the heart upon the ascending part of the azygos, but cross-sections of the thorax (Fig. 3) show this to be impossible. The vein is so completely and

---

[11] Gruber, quoted by McMurrich in Piersol's Human Anatomy, p. 983.

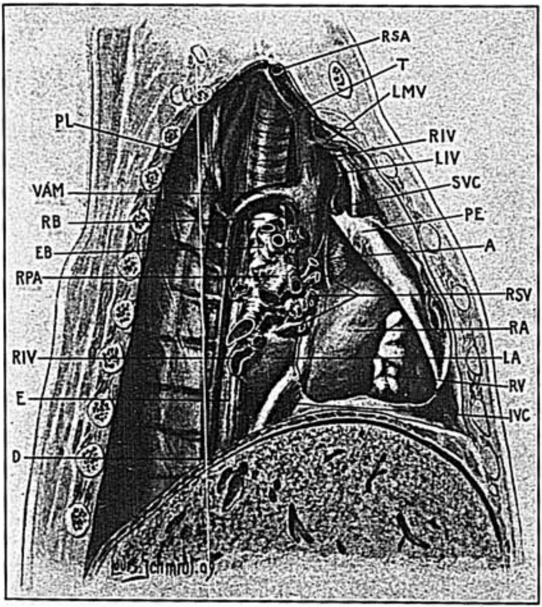

FIG. 1.—View from the right of the mediastinal contents and the root of the right lung. *D*, diaphragm; *E*, œsophagus; *RIV*, right inferior pulmonary vein; *RPA*, right pulmonary artery; *EB*, eparterial bronchus; *VAM*, vena azygos major; *PL*, pleura; *RSA*, right subclavian artery; *T*, trachea; *LMV* (should be *RMV* in the illustration), right internal mammary vein; *RIV*, right innominate vein; *LIV*, left innominate vein; *SVC*, superior vena cava; *PE*, pericardium; *A*, aorta; *RSV*, right superior pulmonary vein; *RA*, right auricle; *LA*, left auricle; *RV*, right ventricle; *IVC*, inferior vena cava.

well protected by the œsophagus and the aorta that such a contingency need not be considered.

Incidentally it might be stated that autopsy reports in which the assertion is made that the azygos major vein is dilated are, as a rule, untrustworthy. One writer (Steele[12]) states that he found this vein dilated "to the size of a goose quill." When one realizes that the normal diameter of this vessel is about 1 cm., it can readily

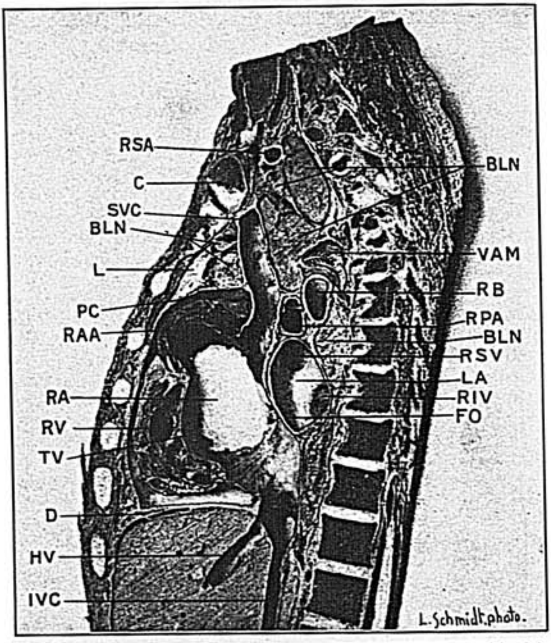

Fig. 2.—Anteroposterior section through the thorax in a plane 2 cm. to the right of the sternum. *IVC*, inferior vena cava; *HV*, hepatic vein; *D*, diaphragm; *TV*, tricuspid valve leaflet; *RV*, right ventricle; *RA*, rignt auricle; *RAA*, right auricular appendix; *PC*, pericardial chamber; *L*, lung; *BLN*, bronchial lymph nodes; *SVC*, superior vena cava; *C*, clavicle; *RSA*, right subclavian artery; *VAM*, vena azygos major; *RB*, right bronchus; *RPA*, right pulmonary artery; *RSV*, right superior pulmonary vein; *LA*, left auricle; *RIV*, right inferior pulmonary vein; *FO*, fossa ovalis.

---

[12] Jour. Amer. Med. Assoc., October 1, 1904.

be seen that such reports are untrustworthy. The average impression is that the azygos major is about the size of a radial artery, and many have been the expressions of amazement when our preparations have been shown to friends and the fact demonstrated that this vessel has at its upper end a normal caliber equal to that of a carotid or even a subclavian artery.

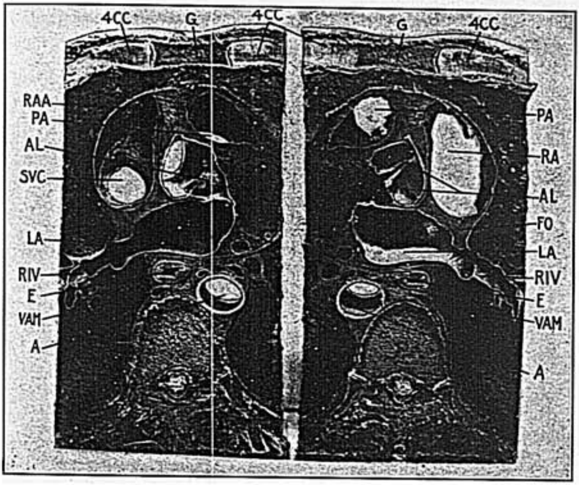

FIG. 3.—Lower (on the left) and upper (on the right) views of a transverse section through the mediastinal portion of the thorax at the level of the fourth costal cartilage. *A*, aorta; *VAM*, vena azygos major; *E*, œsophagus; *RIV*, right inferior pulmonary vein; *LA*, left auricle; *SVC*, superior vena cava; *AL*, aortic leaflets; *PA*, pulmonary artery; *RAA*, right auricular appendix; *4CC*, fourth costal cartilage; *G*, gladiolus; *RA*, right auricle; *FO*, fossa ovalis.

5. Fifth, as to the inability satisfactorily to explain why the effusion is, in a not inconsiderable number of cases, entirely confined to or much greater on the left side. Thus, of 108 cases recorded by Steele and Lord,[13] no less than 30 of them (27.7 per cent.) were left-sided, either entirely or to a great extent. Steele,[14] in his second paper, advanced the theory that this might be explained by the dilatation of the heart being greater on the left side than on the right. "In 11 cases of effusion confined to the left pleura, or greater on that side, the left heart, and especially the left ventricle,

---

[13] Osler's Modern Medicine, vol. iii.

[14] Journal of the American Medical Association, October 1, 1904.

was especially mentioned as enlarged in 9 cases, that is, in four-fifths of the cases of left-sided effusion." He admits, however, that it is difficult to explain these cases on this theory, as there is no large venous trunk analogous to the vena azygos major to be compressed. He also mentions Rosenbach's explanation, namely, that the pressure exerted by the enlarged left ventricle on the left lower lobe produces atelectasis. As a result of long-continued congestion produced by collapse of the lung there arises a low grade inflammation of the pleura, and finally, an effusion forms in the pleural sac which is partly transudate and partly inflammatory. Steele's comment on this is that his own "list of cases gives nothing to confirm or deny the theory of Rosenbach. I can only call attention to the fact that left-sided cardiac dilatation seems to bear some relation to left-sided pleural effusions."

AUTHORS' EXPLANATION. The explanation which we wish to offer to account for these effusions is as follows: The fluid comes not from the parietal, but from the visceral pleura, and the outpouring is caused, so far as the pressure factor is concerned, by dilated portions of the heart pressing on and partly occluding the pulmonary veins.

Two sets of bloodvessels enter the lungs, the bronchial arteries and the pulmonary arteries. The former are nutritional in function and are derived from the aorta or from the first aortic intercostal. They enter the hilum of the lung and follow the posterior surface of the bronchi, some of their terminal branches reaching and supplying the pleura. The pulmonary arteries, in addition to carrying venous blood to the lungs, send twigs to the pleura. In this membrane there is a capillary anastomosis between the terminal branches of the bronchial and pulmonary arteries, on the one hand, and the venous radicles which are tributary, not to the bronchial, but to the pulmonary veins, on the other. This has been shown by Miller,[15] who says: "The capillary network into which the bronchial artery breaks up in the pleura gives rise to radicles which join the pulmonary vein." He also states: "I have failed to demonstrate by the use of granular injection masses any anastomosis between the bronchial and pulmonary arteries. On the other hand, by using injection masses which flow freely I have injected both sets of vessels (pulmonary artery and vein), but only by a backward flow (from the bronchial artery) of the injecting mass through the capillary network." It is evident, therefore, that the venous blood from the visceral pleura is poured into the pulmonary veins, and, as a corollary, that any obstruction to the flow through this vein, if of sufficient power and duration and if accompanied by whatever condition of the blood essential to transudation, would produce leakage through the visceral pleura into the pleural sac.

[15] American Journal of Anatomy, vii, 404, 405.

Points in favor of our theory are that it explains equally well right-sided, left-sided, and bilateral collections of fluid, and also accounts for certain intrapulmonary conditions found clinically and postmortem in association with hydrothorax. Starting with the assumption that the transudate comes from the visceral pleura as a result of pressure on the pulmonary veins, a study of the anatomical relations between the heart and the roots of the lungs illumines the discrepancies which have long been noted. Anatomically the relations and pressure possibilities differ widely on the two sides.

The Anatomy of Right-sided Effusions. These are due to pressure exerted by a dilated right auricle upon the right pulmonary veins and upon the right end of the left auricle. That a dilated right auricle does press upon the right pulmonary veins is shown to be not only possible but inevitable by a study of the mutual relations of the right auricle and the right pulmonary root. It is shown with special clarity if the thoracic contents are hardened in position before the chest is opened. Studies under these conditions were made in three ways, by anteroposterior sections, a valuable method of studying thoracic relations which has been too much neglected, by transverse sections, and by lateral dissections of the mediastinum after removal of the lungs. The following facts were definitely brought out: Of the four chambers of the heart, three appear on the anterior surface and one on the posterior. The anterior cavities are, from right to left, the right auricle (the appendix mainly), the right ventricle, and the left ventricle, and all are plainly visible on anterior inspection. The main cavity or atrium of the left auricle is entirely a posterior structure (Figs. 2, 3, and 4), being invisible from the front, and it comes in relation anteriorly with the three other chambers as well as with the first portion of the aortic arch. Posteriorly it meets firm resistance and is separated from the bodies of the thoracic vertebræ by the contents of the posterior mediastinum, particularly the descending aorta and the œsophagus (Fig. 3). Above it is bounded throughout its entire length by the right pulmonary artery (Figs. 2 and 4), above which is the aortic arch. In front of its right-hand end, into which the right pulmonary veins open, are the right auricle and a little of the superior vena cava (Fig. 2), and in the lower portion of the interauricular wall is the thin fossa ovalis (Figs. 2 and 3), remaining from the foramen ovale.

If the right auricle were to become dilated it could expand forward but slightly, owing to the presence of the chest wall; it could not advance to the left on account of the right ventricle being there; below are the diaphragm and liver; above are the superior vena cava and an overlapping wedge of lung tissue. The path of least resistance is to the right, upward and backward, and in so expanding there would be inevitable compression of the structures which lie behind the right portion of the auricle. These are the right end of the left auricle and the root of the right lung (Figs. 1, 2, and 3). Of the component elements of the latter the most anterior are the pulmonary veins and behind these are the bronchi. The lower portion of the right end of the left auricle extends somewhat below the bronchus, and is bounded posteriorly by the œsophagus and anteriorly by the right auricle. In case, therefore, the latter expands, the two parts of the circulatory system which cannot escape compression are the right pulmonary veins and the right end of the left auricle, compression of the latter being facilitated by the thin fossa ovalis between the two auricles. The result of such compression would be stagnation in the right pulmonary veins and consequent leakage, on the outside, from the visceral pleura, and on the inside, into the lungs, the latter a point to be taken up later.

The Anatomy of Left-sided Effusions. The left auricle is unfortunately named, except in that the name indicates its physiological position. It is the posterior auricle, forming as it does the back surface of the heart, being the only portion of the heart to touch the posterior body wall and lying behind the three other cavities of the heart. It is a slightly flattened, transversely placed cylinder with tributaries entering at both ends (Figs. 2 and 4). From its left upper anterior portion arises its auricular appendix (Fig. 4), which passes forward and curls around the left anterolateral aspect of the root of the pulmonary artery (Fig. 5). Posteriorly the appendix rests against the left upper pulmonary vein and anteriorly it comes close to the chest wall, from which it is separated only by pericardium and pleura and a small thin wedge of lung. Above, outside the pericardium, is the pulmonary artery, below is the left ventricle, and the only direction in which free expansion is possible is toward the left (Fig. 5). The left auricle is so hedged in on all sides that dilatation would first affect the appendix, which must act as a safety valve. The main cavity, or atrium, abuts firmly against the aorta, the œsophagus, and the vertebræ posteriorly (Fig. 3), and marked expansion in this direction would cause dysphagia from pressure on the œsophagus, a symptom not complained of in these conditions. In front are the other chambers of the heart, so placed that enlargement anteriorly would be prevented by the impinging of the anterior cavities against the chest wall. Upward bulging would be blocked by the right pulmonary artery and the aortic arch, and below is the diaphragm, braced and supported by the liver. The natural expansile relief to overfilling would take place first to the left, and this would necessarily mean distention of the auricular appendix.

Occupying a position posterior and to the left of the appendix, separated from it only by the pericardium and the pleura, is the left upper pulmonary vein (Fig. 5). Behind, to the left, and in immediate contact with this vessel is the left bronchus (Fig. 5).

Distention of the left appendix would necessarily squeeze the left upper pulmonary vein between the appendix and the bronchus and be a factor in causing transudation. The left lower pulmonary vein is in its extrapericardiac portion out of reach of the appendix, but it is so placed that dilatation of the left ventricle would involve the vein in compression between the ventricle and the bronchus (Fig. 5). In addition, there is in the left end of the left auricle, a structure which may be a factor in left-sided effusions.

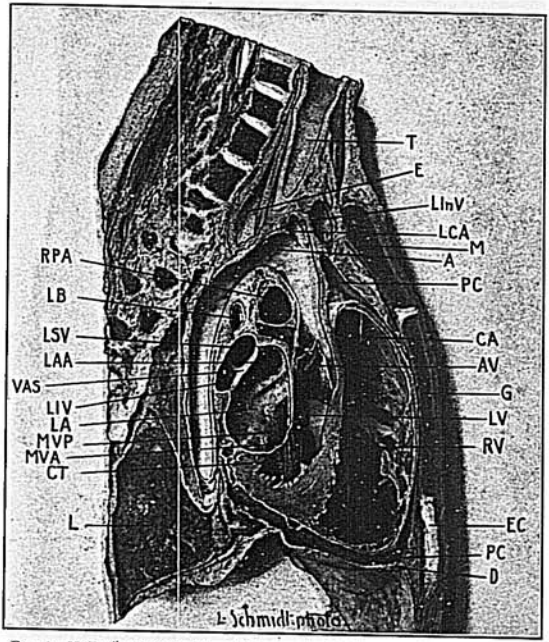

Fig. 4.—Median anteroposterior section through the thorax. *L*, lung; *CT*, chordæ tendineæ; *MVA*, mitral valve, anterior leaflet; *MVP*, mitral valve, posterior leaflet; *LA*, left auricle; *LIV*, left inferior pulmonary vein; *VAS*, veno-appendicular septum; *LAA*, left auricular appendix; *LSV*, left superior pulmonary vein; *LB*, left bronchus; *RPA*, right pulmonary artery; *T*, trachea; *E*, œsophagus; *LInV*, left innominate vein; *LCA*, left common carotid artery; *M*, manubrium; *A*, aorta; *PC*, pericardial chamber; *CA*, conus arteriosus; *AV*, aortic valve leaflets; *G*, gladiolus; *LV*, left ventricle; *RV*, right ventricle; *EC*, ensiform cartilage; *D*, diaphragm.

This is the almost vertical partition, which might be called the veno-appendicular septum, between the opening into the auricle of the appendix anteriorly and the left pulmonary veins posteriorly (Fig. 4). A dilatation of the appendix which would thrust this septum backward would seriously hinder the flow of blood from the left pulmonary veins into the left auricle and be a factor in causing transudation from their radicles.

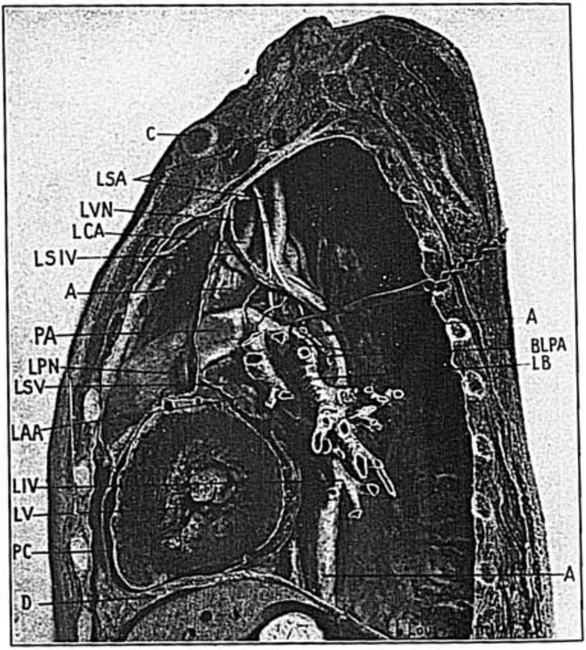

FIG. 5.—View from the left of the mediastinal contents and the root of the left lung. (Retouched photograph.) *D*, diaphragm; *PC*, pericardial chamber; *LV*, left ventricle; *LIV*, left inferior pulmonary vein; *LAA*, left auricular appendix; *LSV*, left superior pulmonary vein; *LPN*, left phrenic nerve; *PA*, pulmonary artery; *A*, aorta; *LSIV*, left superior intercostal vein; *LCA*, left common carotid artery; *LVN*, left vagus nerve; *LSA*, left subclavian artery; *C*, clavicle; *A*, aorta; *BLPA*, branch of left pulmonary artery; *LB*, left bronchus.

Greater frequency on the right side is due to the fact that dilatation of the right auricle is more common and more easy than a similar condition of the left side, and such dilatation is the only factor needed to cause damming back in the right pulmonary veins. On the left side, in order to include both upper and lower veins, there is needed dilatation of the left auricular appendix and of the left ventricle, with possibly a retrodisplacement of the vertical septum mentioned above, three factors as against one on the right side.

As far as we know, but one writer has come any where near the same conclusion as have we. West,[16] in considering the various factors leading to hydrothorax states that "It is not agreed from which set of vessels the effusion comes, but I think it must be from the pulmonary artery or vein; and though the obstruction leads rather to œdema of the lung than to pleuritic effusion, still the two are frequently associated together." Surely, if there is leakage from the pleura there should be a similar filtration into the lung tissue and air spaces, a condition which should cause changes in the breath sounds and the presence of moist rales. If the latter part of West's statement is correct, that is, that œdema of the lung and pleural effusion are frequently associated, it would seem to bear out our theory as to the factors at work. May it not be more than possible that the rales so frequently heard above an effusion and hitherto ascribed to compression of the lung, are due to intrapulmonary leakage from the same pressure on the pulmonary veins that is causing the presence of the fluid in the pleural sac?

Recently one of us had the opportunity of studying a case of failing compensation in the service of Dr. H. A. Hare, in the Jefferson Hospital. There were found signs of a small effusion in the right chest, and the lung above the fluid was the seat of small moist rales. The other lung was absolutely clear. The fact that such cases as this are not uncommon would tend to support the theory advanced in this paper.

A PRACTICAL HOSPITAL POLYGRAPH.

By Theodore B. Barringer, Jr., M.D.,
of New York.

(From the Department of Medicine of Cornell University Medical College, New York.)

A hospital polygraph, in order to be thoroughly practical, must be readily transportable from bed to bed, and should not be so elaborate and heavy in construction as to necessitate moving the patient to it.

---

[16] Diseases of the Organs of Respiration, ii, 757.